Book review

Donovanosis. By V N Sehgal. (Pp 49; Rs 30.) India: Jaypee, 1988.

Almost 35 years ago the latest authoritative and the often quoted monograph on donovanosis by Professors R V Rajam and P N Rangiah was published by the World Health Organisation. During those years several aspects of the condition have remained obscure. They include: the patchy distribution of donovanosis even within tropical countries: the inability to grow the causative agent on artificial media; the low infectivity of the organism; the lack of a reliable serological test; the determinants of systemic spread of the infection; the optimum treatment, especially in pregnancy, in view of the rather poor efficacy of erythromycin alone as observed by some experts; and the length of follow up necessary to ensure cure.

Specialists practising in the areas where the disease is relatively common clearly have a responsibility to carry out research to solve some of the mysteries surrounding this disease. Apart from the availability of several alternative treatment regimens, no significant progress has been made during the past three decades to fill the gaps in our knowledge mentioned above. Professor Sehgal's description of the disease, however, which is mainly factual and largely based on his study of 78 patients, is very well presented, and the subject has been generally updated.

In the differential diagnosis, which Professor Sehgal has discussed in some details, the statement that the "demonstration of Haemophilus ducrevi in Gram stained smear is diagnostic of chancroid" may be considered by some to be untenable. There is also an error in Table 8 in which the "demonstration of Gram negative rods with bipolar condensation of chromatin" is mentioned under chancroid.

There are 37 black and white photographs, most reasonably clear. Unfortunately the intended updating of bibliography is marred by the printer's omission of references 63 to 70. There are helpful appendices at the end with details of the staining techniques for smears and biopsies.

The monograph has 49 pages and is priced at thirty rupees (about £1.25), which seems just about right. I would recommend it to the genitourinary physicians and others who are likely to encounter and required to manage patients with this condition.

O P Arva

Notices

Organisers of meetings who wish to insert notices should send details to the editor (address on the inside front cover) at least eight months before the date of the meeting or six months before the closing date for application.

Hong Kong Genitourinary Medicine Associa-

On 1 October 1987 the Hong Kong Genitourinary Medicine Association was founded at Hong Kong.

The membership of this association is open to all medical staff who are interested in the study of sexually transmitted diseases.

The president is Dr H W Fung, Genitourinary Clinic, 306A Tung Ying Building, 100 Nathan Road, Kowloon, Hong Kong.

Courses on the acquired immune deficiency The fee will be £90, and buffet lunch at the syndrome (AIDS)

The Royal College of Physicians of London is organising courses to train general physicians who will be concerned in the care of patients with AIDS. Each course will last for one week (Mondays to Fridays); mornings will be spent at the College and afternoons at one of four hospitals with major AIDS centres in London (St George's, St Mary's, St Stephen's, and the Middlesex). Numbers on each course will be limited to 20, with groups of five attending each hospital.

college each day and coffee or tea are included.

Starting dates and closing dates for applications are as follows:

Week starting Closing date for 1988 applications 21 November 10 October

For further details and application form, please contact: The Assistant Registrar, Royal College of Physicians, 11 St Andrew's Place, Regent's Park, London NW1 4LE (tel: 01 935 1174).

List of current publications

Selected abstracts and titles from recent reports published worldwide are arranged in the following sections:

Syphilis and other treponematoses
Gonorrhoea
Non-specific genital infection and related disorders
(chlamydial infections; mycoplasmal and
ureaplasmal infections; general)
Pelvic inflammatory disease
Reiter's disease
Trichomoniasis

Candidiasis
Genital herpes
Genital warts
Acquired immune deficiency syndrome
Other sexually transmitted diseases
Genitourinary bacteriology
Public health and social aspects
Miscellaneous

Syphilis and other treponematoses

Unusual central chorioretinitis as the first manifestation of early secondary syphilis

EC de SOUZA, AE JALKH, CL TREMPE, S CUNHA, CL SCHEPENS (Boston, USA). Am J Ophthalmol 1988;105:271-6.

Unusual manifestations of secondary syphilis and abnormal humoral immune response to *Treponema pallidum* antigens in a homosexual man with asymptomatic human immunodeficiency virus infection

JD RADOLF, RP KAPLAN (Dallas, USA). J Am Acad Dermatol 1988;18:423-8.

Treponema pallidum in leukoderma syphiliticum demonstrated by electron microscopy

A POULSEN, L SECHER, T KOBAYASI, K WEISMANN (Copenhagen, Denmark). Acta Derm Venereol (Stockh) 1988;68:102-6.

Intrathecal synthesis of specific IgG in syphilitic patients with human immuno-deficiency virus I infection

F MÜLLER, M MOSKOPHIDIS, H SCHMITZ (Hamburg, Federal Republic of Germany). *J Neurol* 1988;235:252-3.

Enzyme-linked immunofiltration assay for rapid serodiagnosis of syphilis

OE IJSSELMUIDEN, MMHM MEINARDI, JJ van der SLUIS, HE MENKE, E STOLZ, RVW van EIJK (Rotterdam, The Netherlands). Eur J Clin Microbiol 1987;6:281-5.

Gonorrhoea

A 13 year longitudinal analysis of risk factors and clinic visitation patterns of patients with repeated gonorrhea

BF McEVOY, WG le FURGY (Schenectady, USA). Sex Transm Dis 1988;15:40-4.

Spread of Neisseria gonorrhoeae resistant to tetracycline outside the United States of America

MA WAUGH, CJN LACEY, PM HAWKEY, J HERITAGE, A TURNER, AE JEPHCOTT (Leeds, England). Br Med J 1988;296:898.

Host range of the conjugative 25·2—megadalton tetracycline resistance plasmid from Neisseria gonorrhoeae and related species MC ROBERTS, JS KNAPP (Seattle, USA). Antimicrob Agents Chemother 1988;32: 488-91.

Iron uptake from lactoferrin and transferrin by Neisseria gonorrhoeae

WR McKENNA, PA MICKELSEN, PF SPARLING, DW DYER (Chapel Hill, USA). *Infect Immun* 1988:**56**:785–91.

Immunoelectron microscopic localization of outer membrane proteins II on the surface of Neisseria gonorrhoeae

EN ROBINSON, CN CLEMENS, ZA McGEE, JG CANNON (Louisville, USA). *Infect Immun* 1988;**5**6:1003–6.

Multiple protein differences exist between Neisseria gonorrhoeae type 1 and type 4 KW KLIMPEL, VL CLARK (Rochester, USA). Infect Immun 1988;56:808-14.

Prophylaxis of gonococcal and chlamydial ophthalmia neonatorum

M LAGA, FA PLUMMER, P PIOT, et al (Antwerp, Belgium) N Engl J Med 1988;318:653-7.

Randomized comparative study of 0.5 and 1g of cefodizime (HR 221) versus 1g of cefotaxime for acute uncomplicated urogenital gonorrhoea

AH van der WILLIGEN, JHT WAGENVOORT, WO SCHALLA (Rotterdam, The Netherlands). Antimicrob Agents Chemother 1988;32: 426-9.

Non-specific genital infection and related disorders (chlamydial infections)

The role of *Chlamydia trachomatis* in Bartholin's gland abscess

HM SAUL, MB GROSSMAN (Stratford, USA). Am J Obstet Gynecol 1988;158:576-7.

Neonatal chlamydial conjunctivitis. A long term follow-up study

I SANDSTRÖM, I KALLINGS, B MELEN (Stockholm, Sweden). Acta Paediatr Scand 1988;77:207-13.

Comparison of three techniques for detection of *Chlamydia trachomatis* in endocervical specimens from asymptomatic women

J LEFEBVRE, H LAPERRIÈRE, H ROUSSEAU, R MASSÉ (Quebec, Canada). J Clin Microbiol 1988:26:726-31.

Screening for Chlamydia trachomatis in a sexually transmitted disease clinic: comparison of diagnostic tests with clinical and historical risk factors

GP KENT, R HARRISON, SM BERMAN, RA KEENLYSIDE (Atlanta, USA). Sex Transm Dis 1988:15:51-7.

Efficiency and cost effectiveness of field follow-up for patients with *Chlamydia tra*chomatis infection in a sexually transmitted diseases clinic

BP KATZ, CS DANOS, TS QUINN, V CAINE, RB JONES (Indianapolis, USA). Sex Transm Dis 1988;15:11-6.

Serum-specific antibodies for *Chlamydia trachomatis* in premature contractions

I COHEN, E TENENBAUM, M FEIGIN, M ALTARAS, N BEN-ADERET, I SAROV (Beer-sheva, Israel). Am J Obstet Gynecol 1988;158:579–82.

Chlamydia parasitism: ultrastructural characterization of the interaction between the chlamydial cell envelope and the host cell EM PETERSON, LM de la MAZA (Irvine, USA). J Bacteriol 1988;170:1389-92.

Restriction endonuclease analysis of DNA from *Chlamydia trachomatis* biovars

EM PETERSON, LM de la MAZA (Irvine, USA). J Clin Microbiol 1988;26:625-9.

Protective role of magnesium in the neutralization by antibodies of *Chlamydia trachomatis* infectivity

EM PETERSON, G ZHONG, E CARLSON, LM de la MAZA (Irvine, USA). Infect Immun 1988;**56**:885-91.

Non-specific genital infection and related disorders (mycoplasmal and ureaplasmal infections)

Prevalence of Mycoplasma genitalium determined by DNA probe in men with urethritis
TM HOOTON, MC ROBERTS, PL ROBERTS, KK

TM HOOTON, MC ROBERTS, PL ROBERTS, KK HOLMES, WE STAMM, GE KENNY (Seattle, USA). Lancet 1988;i:266-8.

This report attempts to clarify the role of Mycoplasma genitalium as a cause of non-chlamydial non-gonococcal urethritis (NGU). Research has been hindered by difficulties in isolation due to the long incubation period of this organism, and the authors have attempted to bypass these by the use of a whole genomic nick translated

DNA probe. Combined culture and DNA probing was used to detect Mycoplasma hominis and Ureaplasma urealyticum and culture only for Chlamvdia trachomatis and Neisseria gonorrhoeae. The specificity of the probe for M genitalium was checked against 60 different strains of genital and extragenital bacteria, and the sensitivity was estimated at 50-100 pg of DNA per dot-blot. Of 203 men evaluated, 160 (82%) were heterosexual, and these were divided into five groups with: no urethritis, gonococcal urethritis. chlamydial NGU. chlamydial NGU, or persistent or recurrent urethritis. M genitalium was significantly associated only with recurrent urethritis and was not related to any other bacterial isolate. A significantly greater incidence (30%) was found in homosexuals, compared with 11% in heterosexuals. This report seems to affirm the possibility that M genitalium plays a part in the aetiology of some cases of recurrent NGU, but support for the role of M genitalium as an important aetiological agent in acute NGU is lacking. Taylor-Robinson has previously suggested that M genitalium has as its primary habitat the anal canal, with secondary infection of the urethra, and this may explain the greater incidence in the urethras of homosexual men.

D J White

Extragenital *Mycoplasma hominis* infection: a report of two cases

DR SHAW, I LIM (Adelaide, Australia). Med J Aust 1988;148:144-5.

Characterization of tetracycline-resistant strains of *Ureaplasma urealyticum*

JA ROBERTSON, GW STEMKE, SG MACLELLAN, DE TAYLOR (Edmonton, Canada). J Antimicrob Chemother 1988;21:319–32.

Pelvic inflammatory disease

Out-patient treatment of pelvic inflammatory disease with cefoxitin and doxycycline

P WØLNER-HANSSEN, J PAAVONEN, N KIVIAT, M YOUNG, DA ESCHENBACH, KK HOLMES (Seattle, USA). Obstet Gynecol 1988;71:595-600.

Ambulatory treatment of suspected pelvic inflammatory disease with Augmentin, with or without doxycycline

P WØLNER-HANSSEN, J PAAVONEN, N KIVIAT, et al (Seattle, USA). Am J Obstet Gynecol 1988;158:577-9.

Reiter's disease

Evidence that Chlamydia trachomatis causes seronegative arthritis in women

D TAYLOR-ROBINSON, BJ THOMAS, J DIXEY, MF OSBORN, PM FURR, AC KEAT (Harrow, England). Ann Rheum Dis 1988;47:295–9.

Trichomoniasis

In vitro susceptibility of *Trichomonas* vaginalis to metronidazole and treatment outcome in vaginal trichomoniasis

M MÜLLER, JG LOSSICK, TE GORRELL (New York, USA). Sex Transm Dis 1988;15:17-24.

Treatment failure in *Trichomonas vaginalis* infections in females. 1. Concentrations of metronidazole in plasma and vaginal content during normal and high dosage

DHH ROBERTSON, R HEYWORTH, C HARRISON, WHR LUMSDEN (Edinburgh, Scotland). J Antimicrob Chemother 1988;21:373–8.

Cancer after exposure to metronidazole

CM BEARD, KL NOLLER, WM O'FALLON, LT KURLAND, DC DAHLIN (Rochester, USA). Mayo Clin Proc 1988;63:147-53.

We assessed the risk of occurrence of cancer associated with exposure to metronidazole in the 771 female residents of Rochester, Minnesota, who were treated with metronidazole for vaginal trichomoniasis during the period 1960 through 1969 and were followed up for a total of 12,628 person-years. Standardized morbidity and mortality ratios were determined by using an expected number calculated by applying age-specific incidence rates from Rochester studies and Cancer Surveillance, Epidemiology, and End-Results Reporting (SEER) data to the person-years of follow-up. The overall standardized morbidity ratios for cancer at all sites were 1.4 (Rochester, 1978 through 1983), 1.5 (SEER data for Iowa, 1978 through 1981), and 1.2 (SEER data for Connecticut, 1978 through 1981). By site of the cancers, the standardized morbidity ratios greater than unity were those for malignant lesions of the lung, breast, thyroid, bladder, brain, kidney, nasopharynx and oral cavity, as well as for multiple myeloma and malignant melanoma; however, the only significantly elevated standardized morbidity ratio was that for bronchogenic carcinoma. After adjustment for smoking status, the standardized morbidity ratio for bronchogenic cancer was 2·5 (95% confidence interval of 1·3 to 4·4). The standardized mortality ratio for cancer at all sites was 1·4 (95% confidence interval of 0·9 to 2·2). The analysis of these data suggests no significant increase in cancerrelated morbidity or mortality for women exposed to metronidazole for treatment of vaginal trichomoniasis.

Authors' summary

Genital herpes

Use of routine viral cultures at delivery to identify neonates exposed to herpes simplex virus

CG PROBER, PA HENSLEIGH, FD BOUCHER, LL YASUKAWA, DS AU, AM ARVIN (Stanford, USA). N Engl J Med 1988:318:887-91.

Asymptomatic shedding of herpes virus during labor

JW SIMKOVICH, DE SOPER (Portsmouth, USA). Am J Obstet Gynecol 1988;158:588-9.

Humoral antibody response in patients with herpes genitalis: analysis of factors influencing the pattern of disease

JA HALLWORTH, DI STOCKER, D SHARIFF, D SUGRUE, A BUCHAN, GRB SKINNER (Birmingham, England). Med Microbiol Immunol (Berl) 1988;177:145-9.

Comparison of western blot (immunoblot) and glycoprotein G-specific immunodot enzyme assay for detecting antibodies to herpes simplex virus types 1 and 2 in human sera

RL ASHLEY, J MILITONI, F LEE, A NAHMIAS, L COREY (Seattle, USA). J Clin Microbiol 1988;26:662–7.

Differential effect of systemic acyclovir treatment of genital HSV-2 infections on antibody responses to individual HSV-2 proteins

R ASHLEY, K MACK, C CRITCHLOW, M SHURTLEFF, L COREY (Seattle, USA). J Med Virol 1988;24:309-20.

Chronic suppression for six months compared with intermittent lesional therapy of recurrent genital herpes using oral acyclovir: effects on lesions and non-lesional prodromes

SL SACKS, R FOX, P LEVENDUSKY, et al (Vancouver, Canada). Sex Transm Dis 1988;15:58-62.

Dosage and safety of long-term suppressive acyclovir therapy for recurrent genital herpes A MINDEL, A FAHERTY, O CARNEY, G PATOU, M FRERIS, P WILLIAMS (London, England). Lancet 1988:i:926-8.

A double-blind, placebo-controlled trial of the effect of chronically administered oral acyclovir on sperm production in men with frequently recurrent genital herpes

JM DOUGLAS, LG DAVIS, ML REMINGTON, et al (Denver, USA). J Infect Dis 1988;157:588-93

Acyclovir in human breast milk

LJ MEYER, P de MIRANDA, N SHETH, S SPRUANCE (Salt Lake City, USA). Am J Obstet Gynecol 1988;158:586-8.

Genital warts

Genital human papillomavirus infections and cancer

MEMORANDUM FROM A WHO MEETING (Geneva, Switzerland). Bulletin of the World Health Organisation 1987;65:817-27.

Association of coexisting vaginal infections and multiple abusers in female children with genital warts

ME HERMAN-GIDDENS, LT GUTMAN, NL BERSON, THE DUKE CHILD PROTECTION TEAM (Durham, USA). Sex Transm Dis 1988;15:63-7.

Malignant progression of laryngeal papilloma associated with human papilloma virus type 6 (HPV-6) DNA

AP ZAROD, JD RUTHERFORD, G CORBITT (Manchester, England). *J Clin Pathol* 1988;41:280-3.

Advantage of human papillomavirus typing in the clinical evaluation of genitoanal warts: experience with the in situ deoxyribonucleic acid hybridization technique applied on paraffin sections

G von KROGH, SM SYRJÄNEN, KJ SYRJÄNEN (Stockholm, Sweden). *J Am Acad Dermatol* 1988;**18**:495–503.

The predominance of human papillomavirus type 16 in vulvar neoplasia

J BUSCEMA, Z NAGHASHFAR, E SAWADA, R DANIELS, JD WOODRUFF, K SHAH (Baltimore, USA). Obstet Gynecol 1988;71:601-6.

Isolation of a novel human papillomavirus (type 51) from a cervical condyloma

GJ NUOVO, CP CRUM, E-M de VILLIERS, RU LEVINE, SJ SILVERSTEIN (New York, USA). J Virol 1988:62:1452-5.

Value of acetic acid screening for flat genital condylomata in men

RE SCHULTZ, HG SKELTON (Portsmouth, USA). J Urol 1988;139:777-9.

Acquired immune deficiency syndrome

Persistent Campylobacter jejuni infections in patients infected with human immuno-deficiency virus (HIV)

DM PERLMAN, NM AMPEL, RB SCHIFMAN, et al (Denver, USA). Ann Intern Med 1988;108:540-6.

Intestinal infections in patients with the acquired immunodeficiency syndrome (AIDS): etiology and response to therapy PD SMITH, C LANE, VJ GILL, et al (Bethesda, USA). Ann Int Med 1988:108:328-33.

Toxoplasmosis and the acquired immune deficiency syndrome

RE HOLLIMAN (London, England). *J Infect* 1988;16:121-8.

Cryptococcal meningitis in patients with AIDS

WE DISMUKES (Birmingham, USA). J Infect Dis 1988;157:624-8.

Neuropathological changes in an asymptomatic HIV seropositive man

TM LENHARDT, MA SUPER, CA WILEY (San Diego, USA). Ann Neurol 1988;23:209-10.

Evidence for early central nervous system involvement in the acquired immunodeficiency syndrome (AIDS) and other human immunodeficiency virus (HIV) infections: studies with neuropsychologic testing and magnetic resonance imaging

I GRANT, JH ATKINSON, JR HESSELINK, et al (La Jolla, USA). Ann Intern Med 1987;107: 828-36.

In this pilot study the authors' aim was to provide neuropsychological performance data on patients at various stages of human immunodeficiency virus (HIV) related disease. Fifty five ambulant homosexual men were studied, who were either participants in

a longitudinal study of HIV infection, or were patients of the University Hospital, San Diego. They were divided into four groups: group A (n = 15) had the acquired immune deficiency syndrome (AIDS), group B (n = 13) had AIDS related complex (ARC). group C (n = 16) were HIV positive (Centers of Disease Control stages II or III), and group D (n = 11) were HIV seronegative. None of the patients with AIDS was suffering from an exacerbation of his condition or a concurrent infection. The assessments carried out were a battery of neuropsychological tests, a structured psychiatric interview (to be reported later), and, in some, magnetic resonance imaging (MRI).

The neuropsychological tests, which were all previously validated, were the Wechsler adult intelligence scale-revised (WAIS-R)-Vocabulary and WAIS-R-Digit tests, the category test, trail-making test, paced auditory serial addition test (PASAT). logical memory (story learning and recall). visual memory, (figure learning and recall), and symbol-digit paired associate learning tests. MRI was performed on 13/15 in group A and 10/13 in group B only. Blood tests included HIV serology, white blood cell and lymphocyte count, and CD4 and CD8 counts. The cerebrospinal fluid (CSF) was not examined, and patients did not have any other neurological investigations, such as computed tomography or electroencephalography.

The results of the study showed that group A patients were significantly slower in their speed of information processing (PASAT) than all other groups, and worse than group D in abstracting ability (the category test). No other differences in the neuropsychological tests reached statistical significance, though there were a few other trends. Group A patients were worse than group D in logical memory and digit span, and group B and group C patients were worse than group D patients in verbal delayed recall and abstraction. The authors combined the results to identify subjects as being abnormal if at least one test was definitely impaired or at least two tests were probably impaired. By this analysis abnormalities were found in 87% of group A, 54% of group B, 44% of group C, and 9% of group D. The differences were significant (p < 0.001). MRI showed abnormalities in 9/13 group A and 5/10 group B patients, including multiple focal lesions, often subcortical and bilateral, large multiple bilateral abnormalities, sulcal and ventricular enlargement, and in three patients solitary focal abnormalities. When the results of the

MRI and neuropsychological tests were compared, six patients were normal in both, six abnormal in one, and 11 were abnormal on both counts. Drug or alcohol abuse, although common, did not account for the differences.

The authors concluded that patients with AIDS had a high rate of cerebral involvement, even without clinical dementia or gross neurological abnormality. There were, however, no data presented as to whether any of these patients had abnormal neurological signs, although they "had not aroused clinical suspicion of an underlying neurologic disease". Half of the patients with ARC had abnormal results, including those on MRI. The authors felt that the detection of such abnormalities implied that asymptomatic HIV seropositive people may still have neurological impairment. Furthermore, the pattern of abnormality described suggested that in some such patients progression to severe dementia will occur.

R J C Gilson

Diagnosis of *Pneumocystis carinii* pneumonia PD WALZER (Cincinnati, USA). *J Infect Dis* 1988;157:629-32.

Diagnosis of *Pneumocystis carinii* pneumonia: improved detection in sputum with use of monoclonal antibodies

JA KOVACS, VL NG, H MASUR, et al (Bethesda, USA). N Engl J Med 1988;318:589-93.

Human immunodeficiency virus (HIV) infection of the uterine cervix

RJ POMERANTZ, SM de la MONTE, SP DONEGAN, et al (Boston, USA). Ann Intern Med 1988;108:321-7.

Human immunodeficiency virus detected in bowel epithelium from patients with gastrointestinal symptoms

JA NELSON, CA WILEY, C REYNOLDS-KOHLER, CE REESE, W MARGARETTEN, JA LEVY (San Francisco, USA). *Lancet* 1988:i:259–62.

It has been suggested that human immunodeficiency virus (HIV) may cause some gastrointestinal symptoms by a direct effect on the bowel. The authors in this study attempt to substantiate this theory in two ways. Firstly by showing the presence of infectious HIV, and secondly by finding HIV infected epithelial cells in bowel mucosa.

Bowel biopsies were undertaken in 13 HIV infected patients with chronic diarrhoea of unknown aetiology and one seropositive patient with no gastrointestinal symptoms. Most showed acute or chronic inflammation with monocytic infiltration. Four rectal mucosal biopsy specimens were

appropriately prepared and added to a culture of peripheral mononuclear cells (PMC) from seronegative people who had been treated for three days with phytohaemagglutinin. A standard reverse transcriptase assay was used to detect the virus in culture fluids, and the positive fluids were passed to fresh normal mitogen stimulated PMC for production and identification of high reverse transcriptase activity and viral antigen in the cells. Two out of the four specimens gave positive results.

In situ hybridisation using a probe specific for HIV I was performed on a further five rectal, two duodenal, and three colonic mucosal specimens from 10 other patients. Viral nucleic acid was detected in five of the specimens, and two further probes showed similar patterns of hybridisation in the tissues. The nucleic acid was found primarily at the base of the bowel crypts and in the lamina propria in some specimens. By using an argentaffin stain in three of the specimens, the authors were able to show a similar distribution of enterochromaffin cells and HIV infected cells. Not all the enterochromaffin cells gave positive results, however, and other crypt cells showed evidence of HIV infection.

The isolation of infectious virus from rectal mucosa confirms the assumptions from epidemiological data that the bowel is a source of infection. Detection of cells containing HIV in the duodenum implies haematogenous spread to bowel mucosa in addition to direct inoculation. The suggestion that neuroendocrine cells, involved in regulating intestinal motility and function, are the target cells for HIV may be an important step forward in understanding the role of HIV in producing chronic diarrhoea and other gastrointestinal disorders.

A J Robinson

HIV infection in the newborn

S BLANCHE, F VEBER, C ROUZIOUX, et al (Paris, France). Presse Médicale 1988;17:528-32.

Seroprevalence of human immunodeficiency virus among childbearing women: estimation by testing samples of blood from newborns

R HOFF, VP BERARDI, BJ WEIBLEN, L MAHONEY-TROUT, ML MITCHELL, GF GRADY (Jamaica Plain, USA). N Engl J Med 1988;318:525–30.

Heterosexual spread of human immunodeficiency virus in Edinburgh

AJ FRANCE, CA SKIDMORE, JR ROBERTSON, et al (Edinburgh, Scotland). Br Med J 1988;296:526–9.

Prevalence and risk factors of HIV infections among drug users and drug-using prostitutes in Amsterdam

JA van den HOEK, RA COUTINHO, HJA van HAASTRECHT, AW van ZADELHOFF, J GOUDSMIT (Amsterdam, The Netherlands). AIDS 1988;2:55-60.

Seropositivity for HIV and the development of AIDS or AIDS related condition: three year follow up of the San Francisco General Hospital cohort

AR MOSS, P BACCHETTI, D OSMOND, et al (San Francisco, USA). Br Med J 1988;296:745–50.

Sexually transmitted diseases, antibodies to human immunodeficiency virus, and subsequent development of acquired immunodeficiency syndrome. Visitors of homosexual sauna clubs in Copenhagen 1982–1983

B HOFMAN, P KRYGER, NS PEDERSEN, et al (Copenhagen, Denmark). Sex Transm Dis 1988;15:1-4.

Heterosexual transmission of human immunodeficiency virus

AM JOHNSON (London, England). Br Med J 1988;296:1017-20.

Preventing the heterosexual spread of AIDS: are we giving our patients the best advice? N HEARST, SB HULLEY (San Francisco, USA). *JAMA* 1988:**259**:2428-32.

Genital ulceration as a risk factor for human immunodeficiency virus infection

RM GREENBLATT, SA LUKEHART, FA PLUMMER, et al (Baltimore, USA). AIDS 1988;2:47-50.

Investigations of AIDS patients with no previously identified risk factors

KG CASTRO, AR LIFSON, CR WHITE, et al (Atlanta, USA). JAMA 1988;259:1338-42.

Do alternate modes for transmission of human immunodeficiency virus exist?

AR LIFSON (Atlanta, USA). JAMA 1988;**259**:1353–6.

Potential effect of revising the CDC surveillance case definition for AIDS

JK STEHR-GREEN, JM MASON, BL EVATT (Atlanta, USA). Lancet 1988;i:520-1.

Increased risk of suicide in persons with AIDS PM MARZUK, H TIERNEY, K TARDIFF, et al (New York, USA). JAMA 1988;259:1333-7.

Routine preoperative screening for HIV MD HAGEN, KB MEYER, SG PAUKER (Boston, USA). JAMA 1988;259:1357-9.

Human immunodeficiency virus antibody testing: a description of practices and policies at US infectious disease teaching hospitals and Minnesota Hospitals

K HENRY, K WILLENBRING, K CROSSLEY (St Paul, USA). JAMA 1988;259:1819-22.

Absence of antibodies to HIV-2/HTLV-4 in six central African nations

PJ KANKI, J ALLAN, F BARIN, et al (Boston, USA). AIDS Research and Human Retroviruses 1987:3:317-22.

Biologic features of HIV-1 that correlate with virulence in the host

C CHENG-MAYER, D SETO, M TATENO, JA LEVY (San Francisco, USA). Science 1988;240: 80-2.

Bidirectional interactions between human immunodeficiency virus type 1 and cytomegalovirus

PR SKOLNIK, BR KOSLOFF, MS HIRSCH (Boston, USA). J Infect Dis 1988;157:508-14.

Comparison of six serological assays for human immunodeficiency virus antibody detection in developing countries

P van de PERRE, D NZARAMBA, S ALLEN, CH RIGGIN, S SPRECHER-GOLDBERGER, J-P BUTZLER (Kigali, Rwanda). *J Clin Microbiol* 1988;**26**:552–6.

Enzyme immunoassay for detection of human immunodeficiency virus antigens in cell cultures

R VISCIDI, H FARZADEGAN, F LEISTER, ML FRANCISCO, R YOLKEN (Baltimore, USA). *J Clin Microbiol* 1988;26:453-8.

Diagnostic value of specific IgM antibodies in primary HIV infection

JMA LANGE, JV PARRY, F de WOLF, PP MORTIMER, J GOUDSMIT (Amsterdam, The Netherlands). AIDS 1988:2:31-5.

In-vitro production of HIV-specific antibody in children at risk of AIDS

A AMADORI, A de ROSSI, C GIAQUINTO, G FAULKNER-VALLE, F ZACCHELLO, L CHIECO-BIANCHI (Padova, Italy). *Lancet* 1988;i: 852-4.

Antibody-dependent enhancement of human immunodeficiency virus type 1 infection

WE ROBINSON, DC MONTEFIORI, WM MITCHELL (Nashville, USA). Lancet 1988;i:790-4.

Cellular anti-GP 120 cytolytic reactivities in HIV-1 seropositive individuals

KJ WEINHOLD, HK LYERLY, TJ MATTHEWS, et al (Durham, USA). Lancet 1988;i:902-5.

Simultaneous detection of antibody to the human immunodeficiency virus and of the surface antigen of hepatitis B virus in human serum

RS SMITH, SB VIEL, SS FONG, DE PARKS, S WESTRICK, DD RICHMAN (San Diego, USA). J Infect Dis 1988;157:812–6.

Immunological consequences of HIV infection: advantage of being low responder casts doubts on vaccine development

C MARTINEZ-A, MAR MARCOS, A de la HERA, et al (Paris, France). Lancet 1988;i:454-7.

Stages in the progression of HIV infection in chimpanzees

CSAXINGER, HJ ALTER, JW EICHBERG, AS FAUCI, WG ROBEY, RC GALLO (Bethesda, USA). AIDS Research and Human Retroviruses 1987:3:375–85.

Condoms as physical and chemical barriers against human immunodeficiency virus

CAM RIETMEIJER, JW KREBS, PM FEORINO, FN JUDSON (Denver, USA). JAMA 1988:259:1851-3.

Treatment of the acquired immunodeficiency syndrome (AIDS) and AIDS-related complex with a regimen of 3'-azido-2',3'-dideoxythymidine (azidothymidine or zidovudine) and acyclovir: a pilot study

A SURBONE, R YARCHOAN, N McATEE, et al (Bethesda, USA). Ann Int Med 1988:108:534-40.

Effect of zidovudine on serum human immunodeficiency antigen levels in symptom-free subjects

F de WOLF, JMA LANGE, J GOUDSMIT, et al (Amsterdam, The Netherlands). Lancet 1988:i:373-6.

Eighteen homosexual men with persistent human immunodeficiency virus (HIV) antigenaemia who were either without symptoms or had persistent generalised lymphadenopathy (PGL) only were included in this study. They were treated double blind for the first eight weeks in three groups of six. Group A received zidovudine 250 mg every six hours, group B received zidovudine 250 mg and acyclovir 800 mg every six hours, and group C received acyclovir 800 mg every six hours. After eight weeks the code was broken for group C, and these men subsequently went on to take zidovudine 500 mg every six hours for the next four weeks and the other two groups continued receiving their previous regimens. At the end of 12 weeks, the regimens of all three groups were altered and group A received zidovudine 500 mg every 12 hours, group B received zidovudine

500 mg every 12 hours with acyclovir 1600 mg every 12 hours, and group C received zidovudine 500 mg every 12 hours for the following 12 weeks. At the end of 24 weeks the codes for groups A and B were broken. Clinical, haematological, virological, and immunological data were collected weekly in the two weeks before the treatment period and in the first 12 weeks of the treatment period and every 2-4 weeks thereafter. Seven men with persistent HIV antigenaemia from the same cohort who were asymptomatic or had PGL only and did not want to participate in the drug study were compared with the treatment group.

When the results were analysed, in group A when the patients were receiving only zidovudine, five of six men showed a decline in the HIV antigen concentrations and three became HIV antigen seronegative after 1, 20, and 24 weeks of treatment. In group B (who received zidovudine and acyclovir during the trial period) four of the six men became HIV antigen seronegative after 1, 4, 9, and 24 weeks of treatment. Another man in this group became HIV antigen seronegative after one week of treatment, and when the dosage of zidovudine was lowered because of haematological toxicity he showed a rise in HIV antigen to pretreatment concentrations. He became HIV antigen seronegative again with resumption of the previous dose. In group C (acyclovir only during the first eight weeks) there was no significant lowering of HIV antigen concentrations. When the treatment was changed to zidovudine, HIV antigen declined significantly in five men. and two became seronegative after four and 14 weeks. Among the seven not treated, six showed an increase in HIV antigen during follow up and in the seventh HIV antigen concentrations remained stable. A modest improvement in CD4 cell counts was seen in the treatment group compared with the nontreatment group. The haemoglobin concentrations in the treatment group fell, but only two patients required transfusion. There was no serious neutropenia or leucopenia and no important alteration in the total lymphocyte counts in the treatment group. At entry 13/18 men were classified as having Centers of Disease Control (CDC) group III disease, and in nine of them extrainguinal lymph nodes had completely regressed after 24 weeks of treatment.

The authors concluded that in this small series there was no obvious benefit from acyclovir either alone or in combination with zidovudine, accepting that the population was too small for firm conclusions. They have shown the potential of zidovudine in acceptable dosage regimens in the secondary

prevention of more serious HIV related disease and stressed the need for large placebo controlled trials of zidovudine in patients with CDC group II and III disease who are at risk of progressing rapidly to the acquired immune deficiency syndrome.

K Shan

Decline of HIV antigen levels in cerebrospinal fluid during treatment with low-dose zidovudine

J de GANS, JMA LANGE, MMA DERIX, et al (Amsterdam, The Netherlands). AIDS 1988:2:37-40.

Anemia and erythropoiesis in patients with the acquired immunodeficiency syndrome (AIDS) and Kaposi sarcoma treated with zidovudine RE WALKER, RI PARKER, JA KOVACS, et al (Bethesda, USA). Ann Intern Med 1988:108:372-6.

Treatment of cytomegalovirus pneumonitis with foscarnet (trisodium phosphonoformate) in patients with AIDS

C FARTHING, MG ANDERSON, ME ELLIS, BG GAZZARD, AC CHANAS (Edinburgh, Scotland). J Med Virol 1987;22:157–62.

Other sexually transmitted diseases

Urethral infection with *Haemophilus* ducreyi in men

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The treatment of donovanosis (granuloma inguinale)

AS LATIF, PR MASON, E PARAIWA (Harare, Zimbabwe). Sex Transm Dis 1988;15:27-9.

The effect of hepatitis B antigenemia on pregnancy outcome

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SC HADLER (Atlanta, USA). Ann Intern Med 1988;108:457-8.

Genitourinary bacteriology

The effect of a copper intra-uterine contraceptive device on the microbial ecology of the female genital tract

KM ELHAG, AM BAHAR, AA MUBARAK (Safat, Kuwait). J Med Microbiol 1988;25:245-51.

Proline aminopeptidase activity as a rapid diagnostic test to confirm bacterial vaginosis JL THOMASON, SM GELBART, LM WILCOSKI, AK PETERSON, BJ JILLY, PR HAMILTON (Chicago, USA). Obstet Gynecol 1988;71:607-11.

Public health and social aspects

Seroepidemiologic studies on the acquisition of antibodies to cytomegalovirus, herpes simplex virus and human immunodeficiency virus among general hospital patients and those attending a clinic for sexually transmitted diseases

NJ BERRY, DMacD BURNS, G WANNAMETHEE, et al (London, England). J Med Virol 1988;24:385-93.

Miscellaneous

Bread making as a source of vaginal infection with Saccharomyces cerevisiae: report of a case in a woman and apparent transmission to her partner

JD WILSON, BM JONES, GR KINGHORN (Sheffield, England). Sex Transm Dis 1988;15:35-6.

A localized vaginal allergic response in women with recurrent vaginitis

SS WITKIN, J JEREMIAS, WJ LEDGER (New York, USA). J Allergy Clin Immunol 1988;81:412–6.

The role of macrophages and the prostaglandins produced by them in causing the defect in cellular immunity seen in recurrent candidal vaginitis has been shown in previous studies. In this paper the authors attempt to discover the mechanism responsible for the production of macrophage

derived prostaglandin E2 (PGE2). Blood and vaginal wash samples were obtained during an episode of acute vaginitis from premenopausal patients who had no predisposing factors for candidal infections. When paired samples from 64 patients were examined for IgE antibodies to Candida spp, vaginal fluids from 12 women were positive and one woman also had anticandidal IgE in her serum. None of 20 controls had anticandidal IgE. On testing a separate group of 16 patients, who reported symptoms of vaginitis after intercourse, four had detectable concentrations of antiseminal fluid IgE in their vaginal fluids; in three of them the antibodies were not found in the serum. In a separate group of 16 patients who used a diaphragm and spermicides for contraception, eight had vaginal fluid derived IgE to one or more spermicides. When PGE₂ concentrations in vaginal fluids were measured in selected patients, PG was detected only in samples that also contained IgE antibody.

The authors concluded that the occurrence of all these patient groups of IgE antibodies in vaginal fluids, but not in the peripheral circulation, suggests a localised vaginal immediate hypersensitivity reaction. A vaginal allergic response, initiated by candidal or other allergens may predispose to recurrent candidal infections by inducing PGE₂ synthesis that in turn suppresses cell mediated immunity. They envisage that future therapeutic approaches would include

identifying and removing allergens, the use of antihistamines, and possibly even hyposensitisation.

S V Devendra

Clinical features of women with chronic lower abdominal pain and pelvic congestion

RW BEARD, PW REGINALD, J WADSWORTH (London, England). Br J Obstet Gynaecol 1988;95:153-61.

Psychosexual trauma of an abnormal cervical smear

MJ CAMPION, JR BROWN, DJ McCANCE, et al (London, England). Br J Obstet Gynaecol 1988:95:175-81.